ORIGINAL REPORT

Red blood cell phenotype prevalence in blood donors who self-identify as Hispanic

C.A. Sheppard, N.L. Bolen, B. Eades, G. Ochoa-Garay, and M.H. Yazer

Molecular genotyping platforms provide a quick, high-throughput method for identifying red blood cell units for patients on extended phenotype-matching protocols, such as those with sickle cell disease or thalassemia. Most of the antigen prevalence data reported are for non-Hispanic populations. Therefore, this study sought to determine the phenotype prevalence in a single blood center's Hispanic population and to compare those results with previously reported rates in non-Hispanic donor populations. We performed a retrospective review of all serologic and molecular typing from donors who self-reported as Hispanic. The phenotype prevalence was reported and compared with rates from other racial/ethnic groups. A total of 1127 donors who self-identified as Hispanic were screened by serologic methods for Rh and Kell antigens, and 326 were subsequently selected for molecular typing. The most prevalent probable Rh phenotypes were R_1r (26.6%), R_1R_2 (21.5%), and R_1R_1 (20.7%); rr was found in 7.8 percent of donors tested. The percentage of K+ donors in this population was 2.8 percent. The most prevalent Duffy phenotypes were Fy(a+b+) (35.9%), Fy(a+b–) (35.6%), and Fy(a–b+) (27%). Of the donors studied, 15.3 percent had an *FY* GATA mutation. Only 1.5 percent of the donors were Fy(a–b–). The Jk(a+b+) phenotype was found in nearly half of the population. M+N+S+s+ was the most prevalent MNS phenotype from that group, constituting 22.4 percent. A total of 95.7 percent of the donors were Lu(a–b+), and Di(a–b+) was observed in 94.4 percent. The most prevalent Dombrock phenotype was Do(a+b+), constituting 46.9 percent, followed closely by Do(a–b+) at 40.5 percent. Hispanic donor antigen prevalence is distinctly different from other racial/ethnic groups and should be considered when attempting to find extended matched units for these patients. ***Immunohematology* 2017; 33:119–124.**

Key Words: phenotype, Hispanic, blood donors

Molecular testing of donors has become an increasingly common strategy to provide antigen negative units for patients (1) on chronic transfusion protocols at risk for alloimmunization (e.g., patients with sickle cell disease [SCD]), (2) for whom a complete serologic evaluation is labor-intensive and time-consuming (e.g., those with warm autoantibodies), or (3) requiring immunohematologic evaluations that are logistically difficult (e.g., recently transfused patients or those with antibodies for which serologic reagents are not commercially available). Molecular testing of donors has provided a rapid, high-throughput method for predicting the donors' extended phenotypes.

Genotyping the entire donor population is not as yet efficient or economical, however. Requests for antigen-negative units in the United States come primarily for the treatment of patients with SCD who are at risk for alloimmunization. Many blood centers have therefore concentrated their efforts on the recruitment, collection, and genotyping of black donors who are more likely to be antigenically similar to patients with SCD. Thus, many of the reported antigen prevalence rates are from the African American (or black) and Caucasian (or white) populations. There are only a few studies that report antigen prevalence rates for Hispanics.[1,2] According to the 2010 U.S. Census, Hispanics constitute 16.3 percent of the U.S. population—as such, they are the fastest-growing group. Thus, antigen prevalence rates among this group will likely be more necessary in the years to come. This report describes antigen prevalence in a large cohort of donors who self-identify as Hispanic.

Materials and Methods

This is a retrospective study of serologic and molecular testing for red blood cell (RBC) antigens in self-identified Hispanic volunteer blood donors who were tested from 2008 to 2016 at a large regional blood collection center (Virginia Blood Services [VBS], Richmond, VA). VBS has over 80,000 RBC donors annually. VBS donors are asked during registration to self-identify their race/ethnicity, which is recorded on their health history form. The donor is then asked to confirm the accuracy of the form at every subsequent donation. Donors can self-identify as Caucasian/white, African American/black, Asian, American Indian, Middle Eastern/East Indian, Hispanic, or other (for donors who self-identify with more than one choice). There is also an option to leave the race category blank, although fewer than 0.2 percent of donors choose this option. From 2008 to the present, approximately 80 percent of these presenting donors self-identified as Caucasian. In the same time period, donors who self-identified as African American or black constituted 13–14 percent of the total. In 2008, only 1 percent of VBS donors self-identified as Hispanic. In the most recent fiscal year, however, 2.32 percent

of these presenting donors self-identified as Hispanic. A query was run in the VBS Laboratory Information System/Blood Establishment Computer System (LIS/BECS) (Blood Bank Computer Systems, Inc., Auburn, WA) for units from Hispanic donors with historic serologic and molecular testing results. The phenotype prevalence from this query is reported here.

The method for typing donors at the Institute for Transfusion Medicine Clinical Services Immunohematology Reference Laboratory (IRL) in Richmond, Virginia, was as follows (Fig. 1): A daily report was run from the LIS for all donors who presented that day. First-time, self-identified black or group O, D+ or O, D– non-Caucasian donors were selected and screened using a microplate method described in *Immunohematology Methods and Procedures*[3] using Bio-Rad reagents (Dreieich, Germany) for C, E, c, e, and K. Donors were selected for genotyping if their serologic testing indicated them to be K– and R_1R_1, R_2R_2, R_0r, or rr. DNA was extracted using QIAcube (QIAGEN, Hilden, Germany). Genotyping was performed, according to the manufacturer's instructions, using human erythrocyte antigen (HEA) BeadChip DNA array (BioArray/Immucor, Warren, NJ) from 2008 to 2014, and ID CORE XT (Progenika Biopharma/Grifols, Derio, Spain) from 2014 to March 2016. The difference between the two platforms is outside the scope of this report because only antigens tested by both platforms were reported in this study. The only exception is a small number of donors with *RHCE* variants, which were detected by ID Core XT; therefore, we did describe the variants detected by this assay. Rh phenotype prevalence rates were calculated from both the serologic result, for donors who did not qualify for molecular typing (Table 1), and the molecular result for those who qualified (Table 2). If a discrepancy between the serology and the molecular results was identified at the time of testing, it was investigated, resolved, and recorded in the LIS at that time. Thus, the data reviewed in this retrospective study will only include the resolved type. The phenotype prevalence rates from our Hispanic population are reported in Tables 1 and 2 and are compared with those listed in the most recent edition of *The Blood Group Antigens Factsbook*.[4]

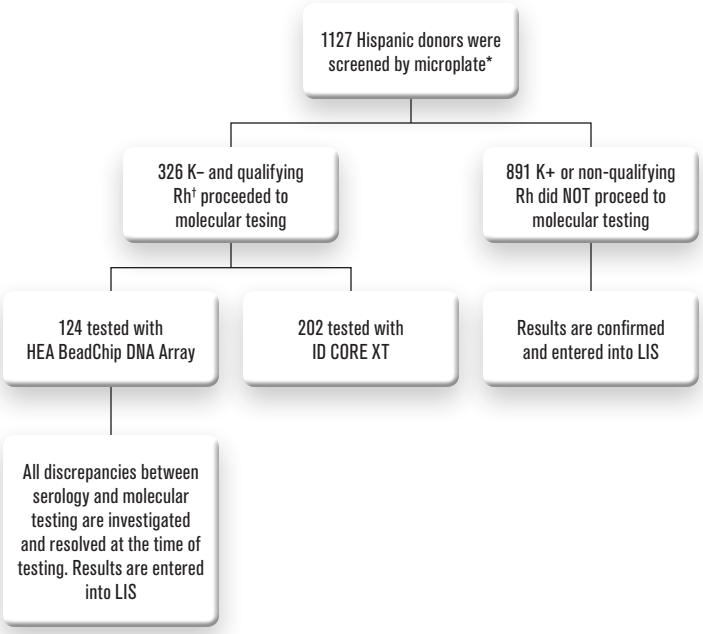

Fig. 1 Method for obtaining historical antigen data. *Microplate screening selection: all first-time, hemoglobin S–negative, black donors, or group O, D+ or O, D– non-Caucasian donors. Initial serologic screening for C, E, c, and K. Donors who screen as C–, E+, and c+ are subsequently tested for e. †Qualifying Rh = R_0r, R_1R_1, R_2R_2, or rr. HEA = human erythrocyte antigen; LIS = laboratory information system.

Results

In total, 1127 unique donors who self-identified as Hispanic were screened by serology and thus had results reported in the LIS/BECS. Molecular testing was performed on 326 of these 1127 donors (those who met the qualifications for molecular typing); specifically, 124 of 326 (38%) donors were typed using BeadChip DNA array (Immucor), and 202 of 326 (62%) were typed using ID CORE XT (Progenika Biopharma).

A total of 1127 donors who self-identified as Hispanic were screened serologically for Rh antigens, and 1125 donors were also screened for K (2 of the 1127 donors were not screened for K for unknown reasons, but likely because of a limited amount of reagent available at the time). The phenotype prevalence of the antigens in the Rh blood group system and for K is listed in Table 1. The k (Cellano) antigen phenotype prevalence was not evaluated because only K– units were selected for molecular testing.

Several Rh variants were identified among the 202 donors tested by the ID CORE XT platform. All Rh variant data were from donors tested using the ID CORE XT (hrB status was not available with the basic BeadChip assay and was thus not further investigated unless required for patient care). In 9 of 202 (4.5%) of these donors, the *RHCE*ce[733G]* allele (c.733G, c.712A, c.1006G) was identified. Thus, the prevalence of V+ in this population also was 4.5 percent. Only 1 of the 202 (0.5%) donors was homozygous for the c.733G single nucleotide polymorphism (SNP) (predicting V+/VS+) associated with loss of the high-prevalence hrB antigen; 8 of 202 (4%) were heterozygous for this variant.

Table 1. Phenotype prevalence for self-identified Hispanic donors compared with other reported races/ethnicities: results from 1127 donors screened by microplate system*

		Percent occurrence†		
Phenotype	**Hispanic N = 1127 (%)**	**Caucasian**	**Black**	**Asian**
R_1R_1 (R_1r')	233 (20.7)	18.5	2.0	51.8
R_2R_2 (R_2r'')	50 (4.4)	2.3	0.2	4.4
R_1r (R_1R_0; R_0r')	300 (26.6)	34.9	21	8.5
R_2r (R_2R_0; R_0r'')	123 (10.9)	11.8	18.6	2.5
R_0r (R_0R_0)	63 (5.6)	2.1	45.8	0.3
R_1R_2 (R_1r''; R_2r'; R_zr; R_0R_z; R_0r^y)	242 (21.5)	13.3	4.0	30.0
R_1R_z	19 (1.7)	0.0	Rare	1.4
R_2R_z	3 (0.3)	0.1	Rare	0.4
rr	88 (7.8)	15.1	6.8	0.1
r'r	7 (0.6)	0.8	Rare	0.1
r''r	2 (0.2)	0.9	Rare	Rare
r'r''	1 (0.08)	0.05	Rare	Rare
V+	9 (4.5)‡	1	30	NA
K+	31 (2.8)§	9	2	NA

*A total of 645 of 1127 donors (57%) were assumed e+ and were not actually tested for e.

†Non-Hispanic percent occurrence retrieved from Reid et al.[4]

‡Only molecular results available from ID CORE XT (*N* = 202).

§*N* = 1125.

NA = not available.

The phenotypic prevalence of Js^a, Kp^a, and antigens in the Duffy, Kidd, MNS, Lutheran, Diego, and Dombrock blood group systems are shown in Table 2. In addition to these results, a total of 15.3 percent of the donors studied had an *FY* GATA mutation. Of these, 9.5 percent were either *FY*B_GATA, FY*A* or *FY*B_GATA* homozygous. Only 1.2 percent (4 of 326) had the point mutation in the *FYB* gene leading to the Fy^x phenotype and conferring weak Fy^b expression; however, 2.1 percent had a single variant allele. In the Kidd blood group system, one donor expressed a *JK*B(IVS5-1A)* variant allele, which is rarely seen except in individuals of Asian or Polynesian descent. In the MNS blood group system, a single donor had a *GYPB*S(IVS5+5T)* variant allele that is commonly seen in the black population. Di(a+) comprised 5.6 percent (18 of 324; 2 results were indeterminate and so not included) of the self-identified Hispanic donors. Finally, 3 percent of these donors had a rare Dombrock allele, although all were heterozygous for this allele. Three (0.9%) donors had a variant *DO*JO* allele; seven (2.1%) had a variant *DO*HY* allele.

Discussion

Although Hispanics make up approximately 16 percent of the U.S. population per recent Census reporting, they constitute about 2.32 percent of our presenting donor population. This is, however, more than a 100 percent increase since 2008. With the demographics of the United States changing rapidly, it is important that we understand the phenotypic prevalence of RBC antigens in this growing demographic. This study was a retrospective review of serologic and molecular testing results from 1127 self-identified Hispanic donors tested between 2008 and March 2016.

The Rh haplotypes for the self-identified Hispanic population are similar to those of Caucasians in that R_1r and R_1R_1 are the most common phenotypes. Additionally, the most common phenotype for African Americans (R_0r), which has been reported to occur in about 45.8 percent of that population, comprises only 5.6 percent of these study donors.[3] The *RHCE*ce[733G]* allele, however, which is common in black (26–40%) and uncommon in Caucasians (<0.01%), is seen in 4.5 percent of the study donors. Because this allele is associated with the VS and V antigens, the prevalence of VS+V+ in this self-identified Hispanic population is also 4.5 percent. The prevalence of V in the Caucasian and black populations is 1 percent and 30 percent, respectively.

We assumed that most donors would be e+, primarily when the donor typed as C+. It is possible that some of these donors are actually e–. For example, of the donors who were typed for e (484 of 1127, 43%), those who were R_1R_2 made up 21.5 percent of the study population, and those who were R_2R_z made up 0.3 percent. This prevalence is similar to that reported in other races/ethnicities with R_1R_2 prevalence

Table 2. Phenotype prevalence for self-identified Hispanic donors compared with other reported races/ethnicities: results from 326 donors by molecular testing

		Percent occurrence*				
Phenotype	**Hispanic, *N* = 326 (%)**	**Caucasian**	**Black**	**Chinese**	**Japanese**	**Thai**
Fy(a+b–)	116 (35.6)	17	9.0	90.8	81.5	69
Fy(a–b+)	88 (27)	34	22	0.3	0.9	3
Fy(a+b+)	117 (35.9)	49	1	8.9	17.6	28
Fy(a–b–)	5 (1.5)	Very rare	68	0	0	0
Fy^b [GATA], Fy^a	26 (8)	NA	NA	NA	NA	NA
Fy^b [GATA], Fy^b	19 (5.8)	NA	NA	NA	NA	NA
Fy^b [GATA] homozygous	5 (1.5)	NA	NA	NA	NA	NA
$Fy^{x\dagger}$	4 (1.2)	NA	NA	NA	NA	NA
	Hispanic	**Caucasian**	**Black**			
Kp(a+b+)	4 (1.2)	2.3	Rare			
Kp(a–b+)	322 (98.8)	97.7	100			
Js(a+b+)	9 (2.8)	Rare	19			
Js(a–b+)	317 (97.2)	100	80			
	Hispanic	**Caucasian**	**Black**	**Asian**		
Jk(a+b–)	87 (26.7)	26.3	51.1	23.2		
Jk(a–b+)	77 (23.6)	23.4	8.1	26.8		
Jk(a+b+)	162 (49.7)	50.3	40.8	49.1		
	Hispanic	**Caucasian**	**Black**			
M+N–S+s–	22 (6.8)	6	2.0			
M+N–S+s+	65 (19.9)	14	7			
M+N–S–s+	40 (12.3)	8	16			
M+N+S+s–	13 (4)	4	2			
M+N+S+s+	73 (22.4)	24	13			
M+N+S–s+	64 (19.6)	22	33			
M–N+S+s–	4 (1.2)	1	2			
M–N+S+s+	13 (4)	6	5			
M–N+S–s+	31 (9.5)	15	19			
M+N–S–s–	1 (0.3)	0	0.4			
	Hispanic	**Most populations**				
Lu(a+b+)	14 (4.3)	7.4				
Lu(a–b+)	312 (95.7)	92.4				
	Hispanic	**Caucasian**	**Black**	**Asian**	**South American Indian**	
Di(a+b+)	18 (5.6)	<0.1	<0.1	10	36	
Di(a–b+)	308 (94.4)	>99.9	>99.9	90	64	
	Hispanic	**Caucasian**	**Black**	**Japanese**	**Thai**	
Do(a+b+)Hy+Jo(a+)	153 (46.9)	49	44	22	13	
Do(a–b+)Hy+Jo(a+)	132 (40.5)	33	45	76.5	86.5	
Do(a+b–)Hy+Jo(a+)	41 (12.6)	18	11	1.5	0.5	

*Non-Hispanic percent occurrence retrieved from Reid et al.[4]

†2.1 percent had the variant Fy^x allele.

NA = not available.

as follows: Caucasian = 13.3 percent, blacks = 4.0 percent, Asians = 30 percent, and R_2R_z prevalence: Caucasian = 0.1 percent, blacks = rare, Asians = 0.4 percent. Because of this assumption, the prevalence of R_1R_2 might be slightly lower, and the prevalence of R_2R_z slightly higher, but we believe the change would be insignificant.

As mentioned earlier, 2.8 percent of this self-identified Hispanic population was K+. This prevalence is similar to

that seen in the black population (2% vs 9% in the Caucasian population). Additionally, in the Kell system, Js(a+) has a population prevalence of less than 0.1 percent in Caucasian individuals and 20 percent in black individuals. We found a prevalence of 2.8 percent in our Hispanic donor population. Kp(a+) was also seen in 1.2 percent of our sample population, compared with 2 percent in Caucasian individuals and less than 0.01 percent in black individuals.

One of the successes of molecular testing was the identification of the FY promoter silencing mutation (67T>C, *FY*01N.01* allele), which is common in blacks but rare in Caucasian populations. This mutation silences expression of Fy^b on RBCs, but Fy^b expression on other tissues is preserved, thus mitigating the need for Fy(b–) RBCs. A recent study by Wilkinson et al.[5] found that 96.8 percent (61 of 63) of patients with SCD had a GATA mutation: 60.3 percent (38 of 63) were FY*02-GATA-67 homozygous (*FY*B_GATA* homozygous), and 36.5 percent (23 of 63) were FY*01/02-GATA-67 heterozygous (*FY*B_GATA, FY*A*). Alternatively, only 17 percent of Caucasians type Fy(a+b–), and far less than 1 percent are Fy(a–b–). Additionally, the GATA mutation is exceedingly rare in Caucasians. In this study population, 15 percent (50 of 326) had the GATA mutation, and over half (52%, 26 of 50) were FY*01/02-GATA-67C heterozygous (*FY*B_GATA, FY*A*). Only 10 percent (5 of 50) were FY*02-GATA-67C homozygous, and 38 percent (19 of 50) were FY*02/02-GATA-67C (*FY*B_GATA, FY*B*). Thus, unlike the black population, most Hispanic donors with an *FY*B_GATA* mutation were also Fy(a+) (52%). This phenotype prevalence was very similar to another study that evaluated phenotypes of Hispanic individuals in South Texas.[1]

Additionally, *FY*B[265T]FY*X,* which results in weak Fy^b expression, is more common in Caucasians than in blacks and was found in four of our donors (1.2%, 4 of 326).

Di^a and Di^b are antithetical antigens. Di^a is a low-prevalence antigen with an occurrence of 0.01 percent in most populations, although it has a much higher prevalence in certain South American Indian groups. It is also reported to occur in 1 percent of Hispanic populations. However, 5.8 percent of our study donors were Di(a+). This is lower than the rate reported by Moulds and Alperin in Mexican Americans living in three Texas communities (14.7%, 8.9%, and 8.2%, respectively).[6]

Homozygosity for *DO*HY* and *DO*JO* [resulting in Hy– or Jo(a–) phenotypes, respectively] are found rarely in the Black population and never in the Caucasian population. We found 1 percent and 1.5 percent of these Hispanic donors had *DO*HY* and *DO*JO* alleles, respectively. Our donors were heterozygous, however. The data on the frequency of heterozygosity for these alleles are not published, to our knowledge.

Using racial self-identification when selecting donors to genotype may efficiently find donors who are more likely to "match" patients—although genetic variability between and even within racial groups is considerable.[7] Self-identification often serves as a proxy for genetic ancestry, but it may not correspond well with genetic ancestry, especially in the Hispanic population, because of the broad definition of the term and reporting biases of the individuals.[8–10] According to the U.S. Office of Management and Budget, the term *Hispanic* used in the 2010 Census "refers to a person of Cuban, Mexican, Puerto Rican, South or Central American, or other Spanish culture regardless of race."[11] As a result of this inclusive definition, a large limitation of this study is whether the data collected from donors who self-identify as Hispanic truly represent a "Hispanic" phenotype, or if one even exists. Additionally, although an individual may acknowledge multiple genetic ancestries, he/she may not identify with and therefore report the corresponding racial group. As such, self-reported ethnicity/race may be an effective tool for screening donors to genotype, but is likely insufficient for mitigating alloimmunization.

Additionally, the Hispanic population constitutes the second largest group of patients affected by SCD in the United States. According to the U.S. Centers for Disease Control and Prevention, "SCD occurs among about 1 out of every 16,300 Hispanic-American births."[12] Extended phenotype-matching transfusion protocols have been used to reduce alloimmunization and the complications of such in black patients with SCD.[13–15] It may also mitigate alloimmunization in the Hispanic population as well.

Finally, RBC genotyping has shown high concordance between molecular and serologic results, and thus it has been proposed that historic molecular results be used to issue units without serologic confirmation.[16] The implementation of this proposal would greatly increase the application of donor typing by molecular methods and thus the practice of genotyping donors.

In conclusion, the U.S. Hispanic population as is currently defined appears to be phenotypically unique in the expression of clinically significant and commonly identified RBC antigens. Additionally, Hispanic donors occasionally express variant alleles seen in other racial/ethnic groups, but typically in different frequencies. Thus, as the U.S. Hispanic population increases, the need for antigenically similar blood will likely continue to increase. This report provides one center's experience with such a population.

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